Utilizing Vitamin A Supplementation to Attenuate Ozone Induced Lung Injury

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Ground level ozone is an air pollutant causing oxidative stress in the lungs, a process capable of damaging lung tissue and exacerbating pre-existing respiratory disorders. Retinoids (vitamin A metabolites) are essential for lung development and adult lung function. The most active retinoid metabolite, all-trans retinoic acid (ATRA), regulates cell differentiation, signaling, and proliferation. Adult lungs expressing lecithin:retinol acyltransferase (Lrat) can accumulate retinyl esters (RE), which are used for ATRA synthesis and have a protective role against lung injury. The purpose of this study is to explore how lung retinoid concentrations impact lung immune response in mouse models of ozone-induced lung injury. Three animal models with different retinoid concentrations were utilized: (1) Lrat+/+ mice fed a chow diet with 15IU/g of vitamin A, (2) Lrat+/+ +A mice supplemented with 400 IU/g of vitamin A, and (3) Lrat-/- unable to store vitamin A. The mice were exposed to air or acute levels of ozone (0.8ppm, 3 hours) and euthanized 24 and 48 hours later. Lung tissue and bronchoalveolar lavage fluid were collected to evaluate lung retinol (ROH) and RE concentrations by high performance liquid chromatography (HPLC). Additionally, mRNA expression of inflammatory (Tnfα, Il-1β and Ptges) and RA responsive genes (Rarγ and Stra6) were measured with qRT-PCR. 48 hours after ozone exposure, significant upregulation in proinflammatory genes were detected in Lrat+/+ mice. Lrat+/+ +A mice showed significantly lower levels of Tnfα, Il-1β, and Ptges in the ozone-exposed lungs. Our results indicate that vitamin A supplementation protects the lungs during acute ozone exposure.